

Nephrol Dial Transplant (1999) 14: 2577–2579

Which diet for diabetic patients with chronic renal failure?

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Introduction

Moderate or severe protein restriction may be proposed in chronic renal failure both to fight its symptoms and to slow its progression [1,2]. Diabetic patients, whether insulin-dependent or non-insulin-dependent, have a chronic disease that has generally existed for a number of years before the occurrence of renal failure. Dietary protein restriction is effective in the progression of diabetic nephropathy [3–5] but several such patients have already been observing dietary recommendations, usually involving carbohydrates and fats for some time and are sometimes unwilling to give up eating habits acquired over a long period. Furthermore, when renal failure develops, the patient may get the impression that the different specialists managing his health have contradictory objectives and give opposing nutritional advice. It is important for the patient not to imagine

that the diabetologist and the nephrologist are giving conflicting dietary directives when, in fact, most of the time their objectives converge.

Nutritional rules for patients with uncomplicated insulin-dependent diabetes

Insulin-dependent diabetes is characterized by a loss of endocrine pancreas function; there is no interference with the peripheral action of insulin. In other words, there is no more, or practically no more production of insulin, although there is no peripheral insulin resistance. Under such conditions, treatment simply consists of insulin therapy, covering the entire circadian cycle. However, this insulin therapy imposes a regularity in food intake and particularly the intake of carbohydrates. Consequently, the patient's nutritional education is limited to teaching him or her how to keep to the same amounts of food and the same carbohydrate rations at each meal. The other dietetic rules con-

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cerning fats and proteins are the same as those of almost everyone, i.e. the attempt to balance the carbohydrate–lipid protein provisions and, of course, an appropriate caloric intake to maintain a stable body weight, as well as to foster the best possible state of health and to preserve the vascular walls which ultimately serves to protect renal function [6].

Dietetic rules for non-insulin-dependent diabetic patients

In these patients, the disease is characterized by disturbances of pancreatic function, sometimes consisting of a lost synchronism of secretion, which is no longer adapted to the level of circulating glucose, but without exhaustion of insulin supply at least early in the course of the disorder. On the other hand, there is an impaired response to the action of insulin, called insulin resistance. This impairment is a hallmark of the disease. The treatment of non-insulin-dependent diabetes consists in trying to achieve a balance between the rate of pancreatic secretion and the degree of insulin sensitivity. The main objective is to lift the inhibition to the action of insulin, since little can be done to restore the faulty pancreatic secretion. The means of going about this mainly involve measures militating against sedentary life style, obesity, and unbalanced diet. Indeed, it has been established that constant excess fat in the diet is a source of impairment to the action of insulin, especially when saturated fats are involved. The dietary educational goals are thus learning to limit the caloric intake and to observe a proper carbohydrate–lipid–protein balance, given that the spontaneous tendency of this population is generally towards the consumption of high amounts of fats and proteins to the almost total exclusion of carbohydrates [6].

Potential impact of dietary measures on chronic renal failure

The protein restriction proposed to patients with chronic renal failure could conceivably be observed in a drastic isolated manner, without replacing protein calories by calories of another source. This would result in an hypocaloric diet and consequently malnutrition. Such a hypothetical therapeutic proposition is, of course, inconceivable.

Protein restriction is in practice carried out by replacing the lost calories with carbohydrate or lipid calories to maintain an adequate caloric intake and ensure a proper state of nutrition. Patients must therefore increase their carbohydrate ration, their lipid ration, or both, to maintain a normal provision of calories.

In principle, whether insulin-treated or not, diabetic patients should be urged to observe a diet defining the amount of carbohydrates and theoretically guaranteeing a balance of carbohydrates, fats and proteins. Because of the reduced protein ration, one has to choose whether to increase the proportion of carbohydrates or that of fats.

What are the potential effects of an increase in the carbohydrate or fat ration in diabetic subject without chronic renal failure?

Studies have shown that both short-term and long-term increases in the carbohydrate ration are accompanied by an improvement in the action of insulin, i.e. a diminution in insulin resistance, which may, however, be offset beyond a threshold of 55% carbohydrates by the development of hypertriglyceridaemia [7].

In contrast, studies of increases in the fat ration, particularly saturated fats, have shown the opposite effect, namely an inhibition of insulin's action. Only Garg *et al.* using monounsaturated or polyunsaturated fats, did not observe a rise in insulin resistance when the lipid ration was increased [8].

In light of this, in chronic renal failure patients without diabetes it is appropriate to compensate the reduction in protein caloric intake by carbohydrate calories. Our experience using diets providing 0.3 g/kg/day of proteins supplemented with ketoanalogues, and in which carbohydrates provide between 60 and 68% of the total caloric intake, have confirmed this premise in chronic renal-failure patients. We have combined studies of insulin sensitivity by euglycaemic hyperinsulinaemic clamp techniques with indirect calorimetry and the use of stable labelled compounds. In this manner we were able to demonstrate that when non-diabetic subjects with chronic renal failure were submitted to protein restriction and the caloric loss was made up by carbohydrate calories, there was a general improvement in insulin sensitivity, improved hepatic sensitivity to insulin, and an amelioration in both oxidative and non-oxidative metabolism of glucose [8–10]. All these results are quite favourable, even for non-diabetic patients, since subjects with chronic renal failure tend to develop an insulin-resistant syndrome leading to hyperinsulinism and ultimately to cardiovascular morbidity.

These observations point to the conclusion that the same reasoning should be used in patients with insulin resistance before the onset of renal failure, which is precisely the case of patients with non-insulin-dependent diabetes. This means that in patients with non-insulin-dependent diabetes, protein restriction and carbohydrate supplementation are nothing less than a realistic medical means of slowing the course of chronic renal failure. Furthermore, whether in presence of chronic renal failure or not this approach should contribute to improving the metabolic stability of these patients.

In patients with insulin-dependent diabetes the proposed management is the same, the consequences being the same except that they lead to a reduction in insulin needs despite the increase in carbohydrate rations as shown in studies by Ciavarella *et al.* [11] and in other studies published by us. The reduction of insulin needs substantiates the effect of the diet upon the peripheral action of insulin but does not always lead to a better control of diabetes, given that a reduction in insulin dose adds to the subtle use of the hormone.

Conclusions

Overall, the diabetic patient who does not suffer from chronic renal failure learns from the diabetologist that the carbohydrate ration must be strictly observed, that quickly absorbed carbohydrates are to be banned, but that complex carbohydrates are vital to the quality of his metabolic stability, that they must be quantified, and above all that they must be regularly provided in sufficient quantity by the diet. The same patient learns from the diabetologist that a high fat ration is harmful for his arterial walls and ultimately for his metabolic equilibrium, especially if he harbours non-insulin-dependent diabetes. When the same patient subsequently sees a nephrologist, after having developed chronic renal failure, the latter will propose a restriction of protein intake adapted to the glomerular filtration rate. As to the general food composition protein restriction is usually associated with a restriction of fat intake and the caloric deficit must be compensated by an increase in carbohydrates. When learning that his usual ration of carbohydrates should increase the patient may lose his bearings with respect to his former eating habits and the initial advice of this doctors. However, this change of dietary advice is not contradictory to previous prescriptions. The increase in the carbohydrate ration, in the absence of changes in total caloric intake, is by no means detrimental to glycaemic control.

Diabetic patients with chronic renal failure should therefore be advised to go on a protein restriction, and this loss in calories should in no case be compensated by fat calories, but by an increase in the amounts of ingested carbohydrates alone. This increase may even exert a beneficial effect on the patient's diabetes. The management of such patients calls for close collaboration between the diabetologist and the nephrologist, so that the patient will not lose confidence in the

doctors and will not believe that the treatment of one disease is being abandoned in favour of another.

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